THE SITE AND FUNCTION OF THE NASAL VALVE.*†

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ABSTRACT.

Previous observers have suggested that the main site of respiratory airflow resistance is localized to the vestibular region of the nose. This resistive segment of the airway was investigated using a "head-out" body plethysmograph in subjects with anatomically normal noses (a) untreated, (b) congested and (c) decongested. In all three conditions, ⅔ of the total nasal airflow resistance was found within the bony cavum in the vicinity of the pyriform aperture and about ⅓ in the cartilaginous vestibule. As might be expected, caval resistance changed proportionately with the degree of mucosal congestion; but, more surprisingly, vestibular resistance changed similarly. This was due in part to the observed forward expansion of the anterior ends of the inferior turbinates with congestion. EMG recordings in subjects breathing through both nostrils demonstrated a gradation of inspiratory alar dilator muscle activity with increased minute ventilation and with mucosal congestion, and there was no evidence of inspiratory alar collapse. But with elevated ventilation through one nostril only, or when the alar muscles were paralyzed by lidocaine block of the VIIth nerve, alar collapse occurred.

These findings are of importance in the management of the congested but anatomically normal nose and in surgery of the nasal tip.

The term nasal "valve" owes its origin to Mink (1920).[1] The word is derived from the Latin "valva" meaning one half of a double folding door. Mink applied the term to the main site of nasal resistance, which he placed at the junction of the upper and lower lateral alar cartilages. We will use the term in a general sense and will present evidence as to its anatomical position and function. Opinions differ both as to the location of the valve and its mode of action. Van Dishoeck,[2,3] employing rigid models and spirometric techniques, placed it at the junction of the upper and lower lateral cartilages, which appeared radiographically as a marked narrowing in the nasal airway. Bridger and Proctor[4,5] measured intranasal pressures with a fine catheter in normal and abnormal noses of subjects breathing through a single nasal cavity. They localized the valve to the region lying between the junction of the upper and lower lateral cartilages and the pyriform aperture and they found that during vigorous inspiration, collapse of this segment brought about flow limitation: it behaved as a Starling resistor.

It seemed important to investigate this subject further since the more recent anatomical studies of Bachmann and Legler[6] have shown the region of the pyriform aperture to be the narrowest part of the anterior nose, and also because previous techniques have entailed considerable departures from normal. Development of a "head-out" body plethysmograph[7,8] enabled nasal airflow to be monitored in a non-invasive manner and avoided the risk of vestibular distortion that attends the use of a face mask. In addition, the plethysmographic technique allowed unrestricted access to the nose for observation and manipulation, for precise positioning of fine intranasal catheters and for simultaneous recordings of EMG's and nasal resistance.

The nose has a higher resistance than that of the remainder of the respiratory airways combined.[9-11] This paper describes the magnitude and location of nasal airflow resistance and the contributions to it of the nasal vestibule and bony cavum, together with the role of the alar muscles in preventing collapse of the former during inspiration. These studies on the site and function of the nasal valve have been presented together in order to portray more clearly the picture of normal nasal respiratory function.

METHODS.

SUBJECTS: Ten Caucasian patients, 6 men and 4 women aged 21 to 60 years, served as subjects. They were selected from among patients with minor complaints of nasal airway obstruction on whom routine airflow studies were performed as a part of their pre and post-therapy documentation. All subjects (except one of the authors) were assessed by the same otolaryngologist and those with marked abnormalities were excluded. Those selected were graded 0-4 with regard to 5 parameters:

Mucosal congestion. This was assessed by anterior and posterior rhinoscopy, by palpation, and by inspection before and after cocainization. "0" represented about 1 mm of congestion and "4" about 3 mm.

Skeletal irregularities. All had more or less straight nasal septums and not unduly prominent turbinates after decongestion, on anterior and posterior rhinoscopy.

Narrowness of vestibules. The width of both the external orifice and the internal os were taken into consideration. The former had a width of about 8 to 12 mm, the latter approximately 2 to 5 mm (at its widest point).

Vestibular narrowing with inspiration. The nose was viewed from below. Narrowing varied from about 0 to 1 mm during a resting inspiration. Subjects whose airway appeared to be completely obliterated during inspiration were excluded.

Ability to flare the nostrils after 10 minutes practice in front of a mirror. Subjects who could not master this maneuver were excluded from the study. Proficiency was graded according to movement of the lateral walls of the vestibule and widening of the external orifice, not with regard to movement of the internal os.

None of the subjects had undergone nasal surgery. The facial nerve block studies were performed on the authors.

*Presented at the 85th Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Palm Beach, FL, May 5, 1982.

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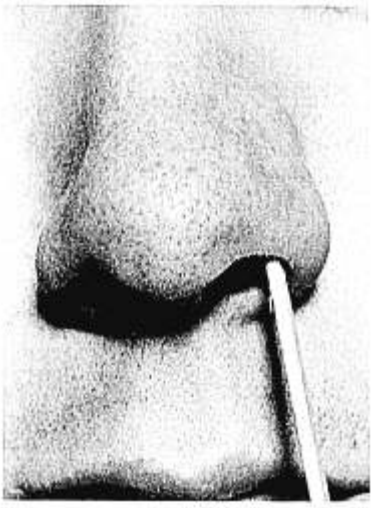

Fig. 1. A subject's left nasal vestibule is opened with a wooden splint, widening is shown in the photograph. Experimentally fine metal probes were used for splinting.

EXPERIMENTAL TECHNIQUES: Each subject was acclimatized for 30 minutes at rest in a comfortably air conditioned laboratory (22-24°C, 50% RH) before resistance testing.

Airflow. The subject to be tested was seated upright in a "head-out" volume displacement body plethysmograph[7] with a large laminar flow element[8] to measure respiratory airflow without facial masking.

Pressure. Transnasal pressure was measured between oropharynx and atmosphere by a differential transducer (Validyne MP45) via a per oral tube (posterior rhinometry).

Intranasal pressure was measured through a plastic catheter (2 mm external diameter) with a lateral opening near the tip. It was passed along the floor of the nose as placing it higher increased its contribution to nasal resistance. It was guided into the inferior meatus to interfere as little as possible with inspiratory airflow which passes primarily through the middle meatus.[3,13] Its depth was measured from an ink mark at the junction of the nasolabial angle with the midpoint of the floor of an external nasal ostium.

Pressure and flow signals were amplified and displayed (Electronics for Medicine DR8 recorder), and amplified output was led also via a buffered interface to an analog-to-digital converter. Pressure and flow signals were sampled every 20 msecs and the digitized values stored in the memory of a programmed microprocessor (Imsai 8080A) which computed and produced a readout and optional print-out of minute ventilation, average transnasal or intranasal pressure and resistance on completion of each series of 5 consecutive breaths.[13,14]

Nasal Splinting. Both nasal alae were opened widely with fine metal probes held manually. Figure 1 shows this being done with wood in one nostril only.

Mucosal congestion was achieved by 3 local applications of 1% histamine acid phosphate to the full length of the inferior and middle turbinates; sneezing ceased and secretion moderated within about 5 minutes leaving the mucosa markedly congested. Airflow resistance gradually decreased to normal over a period of 30-40 minutes.

Mucosal decongestion was obtained by the local application of 0.1% xylometazoline. A stable and long lasting level of decongestion was obtained in 5-10 minutes.

EMG. Skin electrodes were placed near the nasal alar margin and pen recordings made by means of a Beckman recorder.

VIIth nerve block. The subcutaneous facial tissue 2 cm lateral to the nasofacial groove was infiltrated on each side with 5 ml of 2% lidocaine.

EXPERIMENTAL METHODS:

SITE OF RESISTANCE.

Distribution of nasal resistance (6 normal nasal cavities). With 1 nasal cavity occluded (but not distorted) by moist cotton batting, a fine plastic catheter was introduced along the floor of the opposite side and advanced to the nasopharynx. Intranasal resistance measurements were made at 1 cm intervals. The procedure was performed with the mucosa untreated, congested, and decongested. Obstruction of the airway by the catheter was minimized by hooking it around the inferior margin of the nostril and along the nasal floor.

Alar and mucosal components of resistance (10 subjects). In the untreated, congested and decongested nose, resistance to respiratory airflow was determined before and after splinting open of both vestibules. A mean congestive component of resistance was established by repeating measurements because histamine congestion tended to decrease during a protracted testing period.

FUNCTION OF THE VALVE.

Voluntary flaring (8 subjects). In the untreated, congested and decongested nose.

EMG recordings (3 subjects). Electromyographic recordings were made from surface electrodes placed on the skin of the alar margin. These were made at low and elevated minute ventilations, after histamine had been used to induce congestion and, when the nose had been decongested by xylometazoline. Thus, recordings covered a range of nasal resistances between 20 and 1 cm H_2O/l/sec.

VIIth nerve blockade (2 subjects). Nasal patency was measured at sequentially increased minute ventilations after bilateral blockade of facial nerve branches to the alar muscles by injections of 2% lidocaine. The injections were performed well away from the nose so as not to reduce the nasal lumen.

Pressure-flow recording. Transnasal pressure-flow tracings were produced with the DR8 Recorder in the X-Y mode. Photographic records were obtained at resting and increased ventilation with a subject breathing through both nostrils and through one nostril only.

RESULTS.

The ranking of the subjects with regard to the physical criteria detailed under *Methods* is shown in Table I. Two were unable to flare their noses.

SITE OF NASAL RESISTANCE.

Distribution of nasal resistance. Tables II, III and IV show total nasal resistance in the untreated, congested and decongested nose respectively with and without splinting open of the nasal vestibules. It can be seen that opening the vestibular clefts widely reduced the total nasal resistance by about one-third under all three conditions (Fig. 1). This shows that the major resistive segment of the normal nose is not in the cartilaginous vestibule but in the bony cavum whether the nose is congested or not.

These results accord with catheter studies: Figure 2 shows the results from a single nasal cavity: untreated, congested and decongested. They are representative of findings in the other subjects. Caval resistance is almost completely confined to the anterior few millimeters of the bony cavity in the proximity of the anterior end of the inferior turbinate; the relationship of resistance to anatomy is illustrated in Figure 3. Posterior to this resistive seg-

TABLE I.

Clinical Classification of the Ten Subjects Accepted for the Study. All Criteria were Graded on a Scale of 0-4.

Subject	Mucosal Swelling	Skeletal Irregularity	Narrowness Vestibule	Vestibular Narrowing With Inspiration	Adequacy Flaring
1	2	1	3	1	0
2	1	2	1	1	2
3	3	1	3	0	3
4	3	1	2	2	0
5	2	2	2	2	3
6	1	2	1	0	4
7	1	1	1	0	3
8	1	1	2	0	4
9	1	2	3	2	4
10	2	1	1	1	4
Mean	1.7	1.4	1.9	0.9	2.7

ment there was little additional resistance. Anteriorly in the adjacent portion of the cartilaginous vestibule, the remaining one-third of nasal resistance was found.

Alar and mucosal component of resistance. As Tables II, III and IV show and Figure 2 illustrates, airflow resistance within the bony cavum varied proportionately with congestion ($P < 0.005$) as might be expected. More surprisingly, Tables II, III and IV also show proportionately similar changes of resistance in the vestibule ($P < 0.005$). This suggests the presence there of erectile tissue.

We measured the position of the mucosa of the anterior ends of the inferior turbinates relative to a marked point at the nasolabial angle in 6 nasal cavities and found that as the mucosa of the inferior turbinate expanded under the influence of histamine, its anterior tip moved forward by as much as 5 mm in some cases.

Table V shows that the present studies were conducted at low minute ventilations. This was largely because it was often impossible to achieve more in the congested state. Although subjects tended to have higher flow rates when decongested, the inspiratory to expiratory pressure ratios were very similar ($P > 0.1$) suggesting that there was no functionally significant indrawing of the alae in either group. In two subjects, the data was inadvertently erased but was very similar to that in the others.

FUNCTION OF THE "VALVE".

Voluntary Flaring. Two subjects were unable to master the technique (Table I), but the changes in nasal resistance in those able to do so were inconsistent and usually small. Although marked expansion of the alar margin was generally seen, it was not accompanied by consistent widening of the deeper, resistive portion of the vestibule; indeed, in some cases this region narrowed with an increase in resistance (Table VI).

EMG Recordings. Figures 4 and 5 demonstrate that augmented inspiratory alar muscle activity accompanied both a rise in nasal resistance and an increase in nasal minute ventilation. This muscle activity was not seen to bring about alar movement.

Seventh Nerve Blockade. Complete paralysis of voluntary alar movement was not achieved but voluntary flaring was much reduced or absent. Mean

TABLE II.

Nasal Resistance Was Lowered by Splinting ($P < 0.005$) in the Untreated Nose.

Untreated Noses		
Nasal Resistance cms H_2O/litre/sec)		Percent Decrease in Resistance With Splinting
Not Splinted	Splinted	
2.6	1.6	38
2.7	1.7	29
3.7	3.1	16
4.2	1.9	55
3.8	3.6	5
2.2	1.7	23
1.3	0.7	46
3.0	2.8	7
1.9	1.3	32
2.3	1.8	22
Mean 2.77 ± 0.91	2.02 ± 0.88	26.3

TABLE III.

Nasal Resistance Was Increased by Histamine from a Mean of 2.77 to 9.46 cm H_2O/liter/sec ($P < 0.0005$) and Splinting Reduced it by 2.88 cm H_2O/liter/sec ($P < 0.0005$).

Congested Noses		
Nasal Resistance cms H_2O/litre/sec)		Percent Decrease in Resistance With Splinting
Not Splinted	Splinted	
6.8	5.4	21
11.8	6.9	41
17.3	10.7	38
11.3	8.2	27
10.0	7.6	24
8.3	5.2	37
3.5	2.3	34
9.2	7.8	15
5.2	3.4	35
11.2	8.3	26
Mean 9.46 ± 3.88	6.58 ± 2.51	30.8

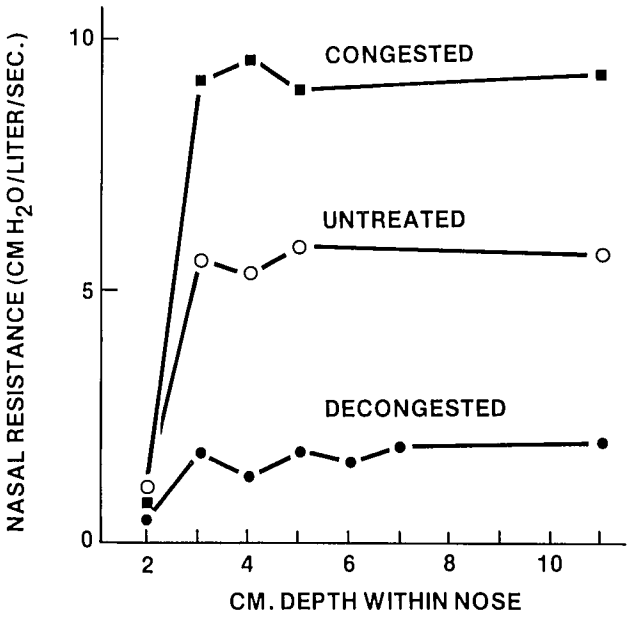

Fig. 2. Respiratory airflow resistance at different depths along the floor of an untreated, congested and decongested nose. In each mucosal state the resistive segment is almost completely confined to a short anterior segment. Depth in cms.

transnasal pressure which normally shows a linear variation with ventilation[13,14] increased exponentially at ventilations greater than 30 l/min. after nerve-block (Fig. 6). This was associated with an increase in inspiratory resistance, the inspiratory: expiratory ratio of transnasal pressure showed a marked increase from a normal of 1.3 to 2.3 indicating a partial alar collapse as the vestibular function changed from flow resistance toward flow limitation.

In the non-paralyzed face, transnasal pressures did not increase exponentially at minute ventilations of 50 l/min. and the inspiratory to expiratory pressure ratios remained about 1.3.

Single Nostril Breathing. Pressure-flow recordings (Fig. 7) showed a tendency toward alar collapse

TABLE IV.

Nasal Resistance Was Reduced by Xylometazoline from a Mean of 2.77 to 1.56 cm H_2O/liter/sec ($P < 0.0005$) and Splinting Decreased it by 0.61 cm H_2O/liter sec ($P < 0.0005$).

Decongested Noses		
Nasal Resistance cms H_2O/litre/sec)		Percent Decrease in Resistance With Splinting
Not Splinted	Splinted	
1.4	0.7	50
2.4	1.7	29
1.6	0.9	44
2.8	1.6	43
1.2	0.7	42
1.1	0.6	45
1.3	0.8	38
1.5	1.0	33
1.4	0.8	43
0.9	0.7	22
Mean 1.56 ± 0.59	0.95 ± 0.39	38.9

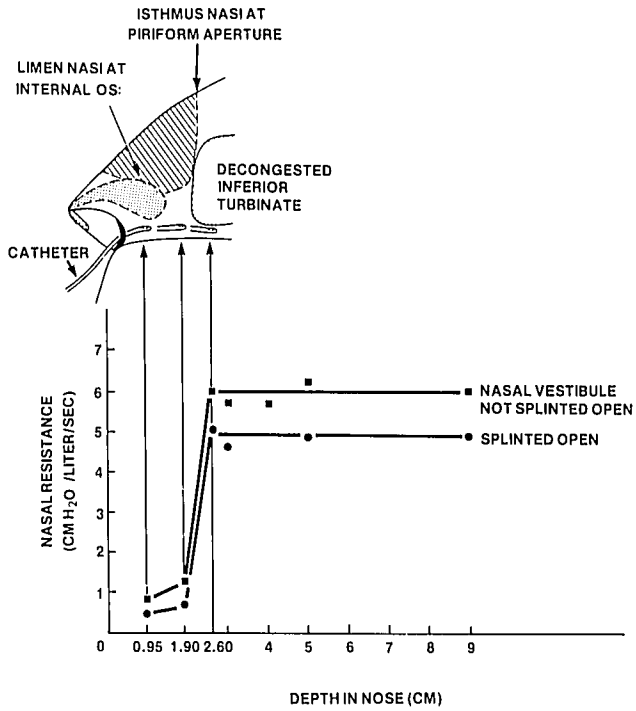
ISTHMUS NASI AT PIRIFORM APERTURE
LIMEN NASI AT INTERNAL OS:
DECONGESTED INFERIOR TURBINATE
CATHETER
NASAL RESISTANCE (CM H2O /LITER/SEC)
NASAL VESTIBULE NOT SPLINTED OPEN
SPLINTED OPEN
0 0.95 1.90 2.60 4 5 6 7 8 9
DEPTH IN NOSE (CM)

Fig. 3. Respiratory airflow resistance at different depths along the floor of an untreated nose. Measurements were made from an ink mark at the mid point of the posterior margin of the nostril. The resistive segment was almost completely confined to the anterior 2.5 cm and the major component was at the isthmus nasi. The effect of splinting is shown.

and flow limitation in a subject breathing through a single nasal cavity as ventilation increased. This was much less marked when breathing through both nostrils at similar minute ventilations.

DISCUSSION.

The Site of the Nasal Valve.

Van Dishoeck[3] stated in 1957 that, "The resistance of an airstream in a short pipe of changing diameters, such as the nose, is mainly determined by the narrowest spot . . ." His radiological studies suggested that this was at the junction of the upper and lower lateral cartilages (limen nasi) but Bachmann and Legler (1972)[6], on the basis of several hundred impressions of the anterior noses of living Caucasian subjects, have shown that the area of the isthmus nasi at the pyriform aperture is less than that of any portion of the nasal vestibule.

Konno (1969, 1977)[15,16] described the major resistive segment in models of the Mongoloid nose and in living subjects as lying anterior to the ostium of the maxillary antrum. Van Dishoeck,[2,3] in his study on casts of the Caucasian nose, localized it to the junction of the upper and lower lateral cartilages. On the other hand, Bridger and Proctor (1970)[4,5] who passed a thin catheter along one nasal cavity while occluding the other in 27 normal subjects, found that this site alone contributed little to nasal resistance and, instead of a solitary resistive locus, there was a progressive increase in resistance over the

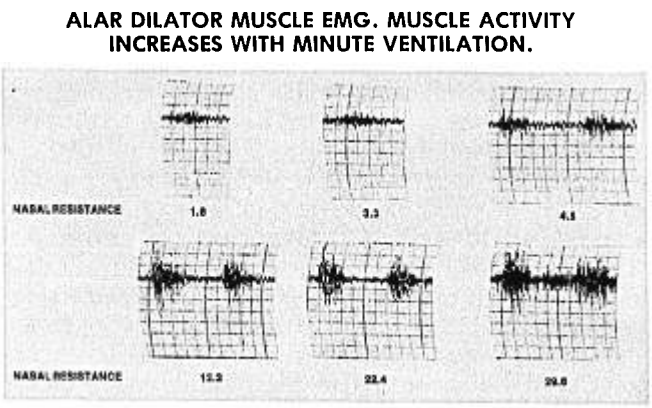

Fig. 4. EMG activity diminished with decreasing nasal resistance during resting ventilation. Resistive changes were induced by local application of histamine and xylometazoline to the nasal mucosa (Minute ventilation: 8.1-10.4).

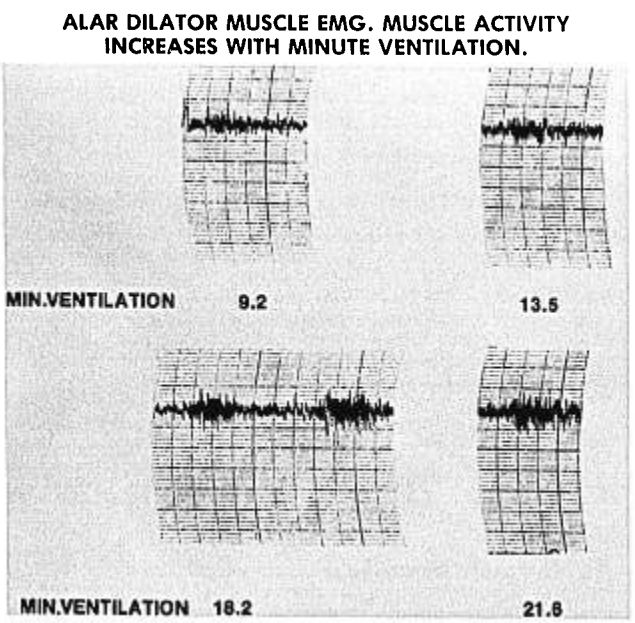

Fig. 5. EMG activity increased with minute ventilation in the untreated nose (Resistance: 2.2-2.6 cm H_2O/liter/sec).

first 2 cm or so of the nose. Beyond this short segment they noted only slight additional resistance, as Van Dishoeck[2,3] had also found. They concluded that resistance was spread over a "flow limiting segment" which corresponded approximately with the length of the upper lateral cartilage. Thus, in the one subject whose data was given,[4] intranasal pressures reached their maximum 1.5 to 2.0 cm from the external nasal orifice. We repeated the observations of Bridger and Proctor[4,5] using a similar technique (Figs. 2 and 3). We passed the catheter along the floor of the nose, to minimize its contribution to nasal resistance, and carefully related its lateral orifice to the intranasal anatomy. Our results confirmed that the "flow limiting segment" contributed to nasal resistance but the greater portion of resistance was situated at the level of the anterior ends of the inferior turbinates within the first few millimeters of the bony cavum. This is graphically illustrated in one subject in Figure 3. Moreover, neither the site nor length of the resistive portion of the nose was affected to any major extent by either a decongestant or a congestant although resistance was altered fourfold (Fig. 2).

Nasal catherization is invasive and causes marked irritation in some subjects. Furthermore, the 2 mm diameter of the catheter is sufficient to increase nasal resistance in the narrow parts of the nose. The method also necessitates blocking the opposite nostril which interferes with normal nasal breathing. Therefore, we also studied subjects breathing through both sides of their nose, by the use of a "head-out" body plethysomograph, and separated the resistance of the bony cavum from that of the vestibular "flow limiting segment" by splinting the latter widely open (Fig. 1). We found that, after minimizing vestibular resistance by this means (Fig. 3), total nasal resistance remained about two-thirds of the previous value in the untreated, congested and decongested nose (Tables II, IV and IV). In other words, the main site of resistance in both the decongested and congested nose lies just within the bony cavum. As one would expect, the resistance of this segment demonstrated considerable variability: from 1.0 cm H_2O/1/sec after xylometazoline (Table IV) to 6.6 cm H_2O/1/sec after histamine (Table III).

More surprisingly, we found that resistance within

TABLE V.

The Same Inspiratory:Expiratory Pressure Ratio of 1.3 in Both the Congested and Decongested Nose Shows that High Transnasal Pressures in the Congested Nose Do Not Increase the Tendency Toward Alar Collapse. It Suggests that the Transmural Pressures are Exerted on Non-Compliant Tissues.

	Congested Noses			Decongested Noses		
Subject	Nasal Resistance	Inspiratory to Expiratory Average Pressure Ratio	Ventilation Liter/Min	Nasal Resistance	Inspiratory to Expiratory Average Pressure Ratio	Ventilation Liters/Min
1	6.8	1.2	9.4	1.4	1.3	13.1
2	11.8	1.4	3.6	2.7	1.4	12.0
3	17.3	1.3	3.4	1.6	0.8	12.1
4	11.3	1.5	10.7	2.8	1.5	20.3
5	10.0	—	—	1.2	—	—
6	8.3	1.1	10.0	1.1	1.0	10.4
7	3.5	1.3	11.5	1.5	1.5	12.6
8	9.2	—	—	1.5	—	—
9	5.2	1.3	7.2	1.4	1.0	7.9
10	11.2	1.0	3.9	0.9	1.4	15.4
Mean	9.43 ± 3.88	1.26 ± 0.16	7.46 ± 3.41	1.68 ± 0.59	1.24 ± 0.27	12.98 ± 3.66

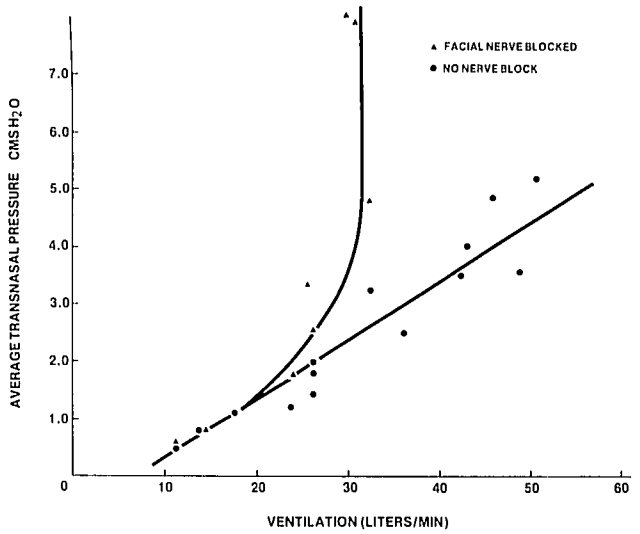

Fig. 6. Average transnasal pressure varied in a linear manner with minute ventilation in the normal nose. Flow limitation occurred at 30 liters/minute ventilation with bilateral alar muscle paresis.

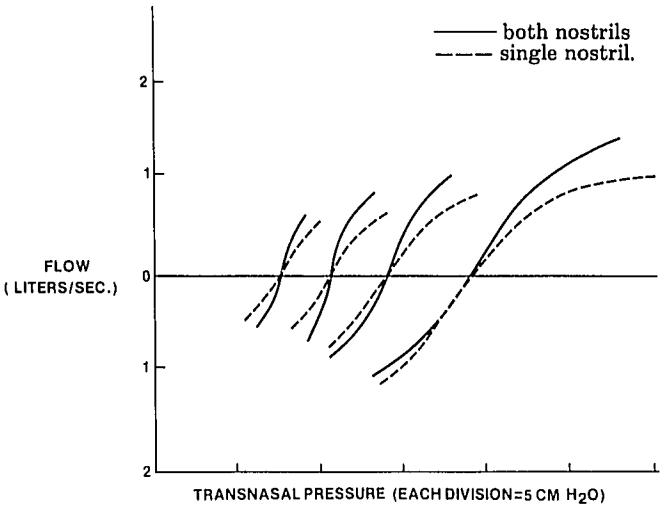

Fig. 7. Transnasal pressure-flow tracings with increasing ventilations. All inspiratory curves exhibited an asymptotic trend at maximum flow but this was more marked in single nostril recordings, in which inspiratory flow limitation developed at the highest ventilation. Inspiration is displayed above the abscissa and expiration below it.

the cartilaginous nasal vestibule also changed in similar proportion to the cavum with mucosal congestion. Tables IV and III demonstrate that splinting reduced total nasal resistance by 0.6 cm H_2O/l/sec after xylometazoline but by 2.9 cm H_2O/l/sec after histamine. Clearly some of the mucosa in the vestibule had erectile properties and our measurements have shown that after the application of histamine, the anterior ends of the inferior turbinates advanced by as much as 5 mm in some subjects. A further contribution may be made by the mucosa of the septum and lateral walls whose histological appearance suggests that they are capable of becoming engorged with blood.[17,18] Because of the narrowness of the region, minor increases in mucosal volume would be expected, by Poiseuille's law, to induce pronounced resistive changes.

Function of Valve.

Bridger and Proctor[4,5] found that when subjects inhaled through one nostril only, partial collapse of the "flow limiting segment" occurred at ventilations of about 30 litres/minute, preventing increases in the intranasal pressure from further augmenting flow (Starling valve effect). We repeated this experiment and obtained comparable results (Fig. 7). However, when the subject was breathing through both nostrils, airflow was not limited and pressure did not increase exponentially in the non-paralyzed face. The difference between the unilateral and bilateral results is partially explained by the lower transnasal and transmural pressure gradients attained when breathing through both nostrils.

In this latter case, the tendency of the compliant vestibule to respond to transmural inspiratory pressures by collapse of the alar walls against the septum is resisted by the phasic activity of the alar muscles and vestibular patency is maintained. Figures 4 and 5 show a graded muscle response which increases with minute ventilation and with nasal congestion. By contrast, when the facial nerve was blocked bilaterally with local anesthetic, inspiratory vestibular narrowing and a marked increase of resistance was found at ventilations above 30 l/minute (Figure 6). Van Dishoeck[2,3] also demonstrated unilateral inspiratory alar collapse in a patient with a facial palsy, and confirmatory photographic illustrations have appeared in the literature.[19,20]

Voluntary flaring of the alae might be expected to reduce vestibular resistance in much the same way as splinting and some physicians recommend flaring exercises as a means of treating "valve problems." Nevertheless, we found that maximal flaring did not consistently lower nasal resistance (Table VI). Indeed, the medially placed origins of the alar muscles[21-25] (levator labii superioris alaeque nasi, dilator naris or nasalis, and the putative muscular dilatator of Sappey)[26] which are inserted into the lower lateral crura distal to the "flow limiting segment,"[22,23] argue against a major dilator action. Levator alae nasi and nasalis take origin above and below the

TABLE VI.

The Changes in Nasal Resistance with Flaring Were Not Consistent, and in Some Cases Were Very Small.

Subject	Untreated (%)	Congested (%)	Decongested (%)
1	—	—	—
2	+46	−11	+11
3	−35	−26	−31
4	—	—	—
5	− 5	− 1	0
6	+ 9	+ 1	−10
7	0	−11	0
8	+10	+ 5	+13
9	− 5	− 4	−21
10	+13	−27	+11

lateral crura respectively so that, when acting together, they would be expected to augment alar rigidity and resist the collapse which might otherwise occur. On the other hand, flaring may result from the unopposed action of the levator alae nasi producing visible rotation of the lower lateral cartilage. Thus, flaring would seem to be mainly a movement of facial expression, but the graded inspiratory contraction (Figs. 4 and 5) of the alar muscles is important in preventing vestibular closure at high inspiratory flow rates or when the nose is congested. It probably arises with the common medullary outflow to the inspiratory musculature. The load on the alar muscles is lightened both by the normal distribution of resistance ($^1/_3$ in the vestibule, $^2/_3$ in the cavum) which ensures that the greater transmural pressures are exerted on the noncompliant bony walls of the cavum and by the fact that at minute ventilations greater than 35 l/minute, there is a switch to mouth breathing which halves transnasal airflow and pressure.[27,28]

CONCLUSION.

From our results it may be concluded that the main airflow resistance of the whole respiratory tract[9-11] is normally confined to a short nasal segment of a few millimeters which is situated in close proximity with the junction of the compliant cartilaginous vestibule with the rigid bony cavity of the nose. The vestibule itself contributes only one-third to this resistance and the resistance of both vestibule and cavum are increased by mucosal congestion. The patency of the vestibule is maintained against transmural inspiratory pressures by structural alar rigidity and phasic alar muscle activity.

In addition to their physiological interest, these findings have important implications for the treatment of congested but anatomically normal noses and for rhinoplasty. In the former, reduction of the swollen anterior ends of the inferior turbinates is of paramount importance. In the latter, it is essential to protect the structural integrity of the cartilaginous framework and the attachments of the alar dilator muscles since weakening of these structures could lead to inspiratory alar collapse and airflow limitation.[4,29,30]

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